CME

The Historic Origins of Military and Veteran Mental Health Stigma

and the Stress Injury Model as a Means to Reduce It

CME EDUCATIONAL OBJECTIVES

1. Restate the historic origins of mental health stigma in the military.
2. List the causes of glutamate neuron apoptosis or necrosis that have been documented in preclinical studies, which also may be relevant to clinical mental disorders in service members and veterans.
3. Explain how stigma may be reduced by conceiving of severe, persistent distress, or functional impairment as literal injuries to the brain and mind.

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Dr. Nash; Ms. Silva; and Dr. Litz have disclosed no relevant financial relationships.

The opinions and assertions contained in this paper are the private views of the authors and do not necessarily reflect the official policies or positions of the Department of Defense or the Department of Veterans Affairs.

doi: 10.3928/00485713-20090728-05

One of the greatest obstacles to the early and effective treatment of mental disorders arising from the stress of military operations is the heavy burden of social stigma associated with them. Military service members avoid seeking care for mental health problems because they are afraid they will be branded as weak or lose the respect and trust of their peers and leaders.[1] Additionally, mental health stigma causes individuals suffering from mental disorders to lose respect for themselves, whether or not they receive treatment. Shame arising from stigma worsens depressive symptoms and social alienation and increases rates of treatment noncompliance and drop-out.[2]

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Of course, mental health stigma is not unique to military and veteran populations, and service members and veterans are particularly vulnerable to stigma for several reasons. Military service members and veterans are at greater risk than most civilians for developing posttraumatic stress disorder (PTSD) and other stress-related mental disorders. Military culture may have little tolerance for weakness, whether physical, mental, or moral. Particularly during wartime, young men and women volunteer to join the military partly because they want to prove themselves in the field of battle.[3] To the extent combat is conceived to be a personal test, the negative impact on self-concept and self-esteem of perceived failure may be great. Furthermore, conceptions of mental disorders as emblematic of personal weakness that have fueled the prejudices of social stigma were born and nurtured in military services in wartime.

Reducing mental health stigma among military service members and veterans, and the institutions that support them, is imperative. But what are the sources of stigma over which clinicians have direct control? How can healthcare providers reduce the burden of stigma for their patients and families during routine clinical encounters? This article answers those questions by reviewing the history of stress-induced mental disorder conceptions that have contributed to stigma in the military. The article also describes an alternative model that reduces stigma by conceiving of such disorders as literal injuries to the brain and mind that are no more the fault of the individual, or a sign of personal weakness, than any other combat wound. The implications of this model for clinical care and prevention will be briefly discussed.

THE RISE AND FALL OF MEDICALIZATION OF COMBAT STRESS AND PTSD

Characteristic features of combat stress and PTSD are clearly evident in ancient dramatic narratives of war and its aftermath. Homer's *Iliad* and *Odyssey,* respectively chronicling the experiences of Achilles and Odysseus during and after the Trojan War, contain compelling descriptions of lasting combat-related distress and functional impairment very much like the modern construct of PTSD.[4,5] One of the oldest surviving plays by Sophocles tells the story of the hero Ajax's suicide in disgrace after brutally slaughtering farm animals in a dissociative flashback to the Trojan War.[6] However, mental distress or impairment during or after combat were not believed, in antiquity, to represent symptoms of an injury or illness, but rather the result of direct interventions by gods over whom mere mortals were powerless.[7] It was not until approximately eight centuries after the mythical Trojan War that the father of modern medicine, Hippocrates, declared that madness, like epilepsy, could be caused by a diseased brain.[8]

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This insight by Hippocrates was slow to gain acceptance. Over the ensuing 2,000 years, madness, both on and off the battlefield, was mostly blamed on supernatural forces. The first recorded description of a syndrome of disabling mood and anxiety symptoms occurring in soldiers at war was that labeled "nostalgia" by military surgeons in 18th century.[9] During the Age of Enlightenment, the scientific method was beginning to be used as a means to understand the entire natural world, including the behavior of humans under stress. The significance of this scientific quantum leap can hardly be overestimated. For the first time, failures to adapt to overwhelming wartime stressors were seen as symptoms that deserved to be treated, and military commanders were given a humane alternative to execution for cowardice.

During one of the bloodiest wars of all time, the American Civil War, combat stress casualties were given diagnostic labels betraying an increasingly medical model, such as "soldiers' heart," "irritable heart," and "sunstroke."[10] Medical conceptualization of stress and stress disorders reached its zenith during the early years of World War I, when "shell shock" was believed to be entirely due to physical disruption of the brain caused by a nearby artillery blast. The assertion during the 19th and early 20th centuries that combat stress — and its contemporary civilian counterpart, "railway spine" — were physical ailments that deserved medical treatment also implied that military stress casualties should be evacuated from war zones, and that both military and civilian trauma victims should receive disability compensation if they didn't recover. By 1916, epidemics of shell shock among French and British troops, and "nervenshock" among the Germans, had drained treasuries and manpower pools on both sides of the war. In this context, skepticism about the physical nature of traumatic stress also grew steadily. After all, no evidence of literal brain damage had ever been found in shell-shocked veterans, and many of them were discovered to have never been near an explosive blast. Furthermore, suggestive therapies

like hypnosis and coercive therapies like electric shock treatment often caused at least the observable somatic manifestations of stress disorders to remit.

To address the nervenshock crisis, the German Association for Psychiatry convened a special "War Congress" in Munich on September 21, 1916.[11] After briefly discussing the evidence on both sides of the debate, the leading psychiatrists and neurologists of the day (then mostly Austrian or German) settled the debate by voting that persistent distress or functional impairment following exposure to a traumatic stressor could only occur in an individual with "hysteria," a pre-existing personality weakness. Subsequently, the German government was relieved of its responsibility to pay disability pensions to veterans suffering from combat stress, and commanders in the field no longer had to evacuate stress casualties away from the front. The French, British, and later, the Americans, adopted an almost identical doctrine and policy. The term "shellshock" was banished from the military medical nomenclature and the principles of "forward psychiatry" were promoted as a means to prevent epidemics and restore combat stress casualties to combat duties.

The term "hysteria" was never intended to be a neutral descriptor. It was chosen to be intentionally stigmatizing, especially when applied to male service members who understood it to be a feminizing term.[11] The demedicalized model adopted in 1916 and the barrier of shame it erected between stress symptoms and their recognition and treatment helped reduce the rates of wartime psychiatric evacuations during the 20th century from approximately 10% in WWII and 3.7% in the Korean War to barely 1.2% during the Vietnam conflict.[12] However, as evident in the mental health burden borne by Vietnam veterans after the war, the demedicalized model failed to prevent long-term disability or to encourage those afflicted to seek treatment.

Both the *Diagnostic and Statistical Manual of Mental Disorders,* first edition (DSM-I), in 1952[13] and DSM-II in 1968[14] included the term "hysteria," both as a synonym for "over-reactive" and as the presumed etiology for a variety of somatic, anxiety, and dissociative disorders. Partly in backlash to this continued stigmatization, DSM-III in 1980 not only removed all references to hysteria but described the new diagnostic entity of PTSD as the direct result of a stressor event "that would evoke significant symptoms of distress in almost everyone."[15] By encouraging the conception of PTSD as "a normal reaction to an abnormal event," as it has often been called since the 1980s, DSM-III signaled the final shift away from a medical model in favor of a normalization model. This new model reduced the stigma associated with acute distress or loss of function in the immediate aftermath of a potentially traumatic event or loss but perhaps at the expense of the stigma associated with stress symptoms that fail to quickly resolve. Regardless of how many times service members and veterans were told that traumatic stress symptoms were "normal," they knew that only a minority of those exposed to even the most horrific events developed clinically significant PTSD. What else but personal weakness could account for failing to recover from something that others seemed to shrug off? DSM-IV added to the criteria for PTSD a requirement for peritraumatic terror, horror, or helplessness, softening the normalization rhetoric by recognizing that the impact of a stressor event on the individual was as important as the nature of the event, itself, or the individual's pre-existing vulnerabilities.[16]

WHAT'S WRONG WITH A DEMEDICALIZED VIEW OF COMBAT STRESS?

The conceptions of combat stress and PTSD as entirely due to either pre-existing personal weakness or merely normal reactions to abnormal events are problematic because: a) they don't fit all the facts, and b) they sometimes do more harm than good. Massive and expensive attempts during WWII to screen out the mentally vulnerable before induction failed to prevent large numbers of combat stress casualties in all military services and all theaters of war. A number of those who failed preinduction psychiatric screening yet managed to join the military anyway became bona fide war heroes. Studies by the Army during the Italian campaign in 1944 confirmed what had long been apparent — after enough days of constant exposure to intense combat, everyone became a psychiatric casualty. The breaking point for most came after about 210 days of continuous combat exposure.[17] Studies of PTSD risk and resilience factors have shown that postexposure social factors are at least as important in determining outcome as pre-existing biological or psychological vulnerabilities. A recent meta-analysis of 2,647 studies of PTSD found that peritraumatic psychological processes were stronger predictors of chronic PTSD than were pretrauma or posttrauma factors.[18]

By silencing debate on the etiology of what is now known as PTSD, the psychiatrists and neurologists who attended the German War Congress in 1916 also stifled other scientific inquiries into the nature of stress disorders in the military. Now, almost a century later, the literature on combat PTSD is comprised almost entirely of retrospective cross-sectional studies rather than the prospective, longitudinal research needed to determine the role really played by pre-existing vulnerabilities. The normalization model has also discouraged those most directly responsible for preserving the psychological health of service members — military leaders — from investing in more effective prevention and early intervention programs. After all, if all combat stress is normal, what is there to prevent or treat?

The greatest potential harm from the view of combat stress as either due to personal weakness or merely normal may be the blame these conceptions place on the individual sufferer. Implied in these models is the belief that each individual chooses, at some level, whether to be strong, tough, and resilient — even choosing whether to have stress symptoms. The 1996 *War Psychiatry* textbook warned that "most psychiatric casualties unconsciously seek a medical exit from combat," and may mimic legitimate medical symptoms in order to save face.[19] Military resilience training based on the current "Battlemind" program teaches that the "inner strength" that enables soldiers to courageously face adversity in combat will also empower them to overcome the readjustment difficulties and stress symptoms they experience after deployment.[20] Implied in this otherwise encouraging and normalizing training is the pernicious message that a failure either to withstand adversity in a war zone or to recover quickly and completely from postdeployment PTSD symptoms may be due to a deficit in "inner strength" or willpower. A popular speaker on the subject of combat stress in both military and law enforcement settings, Dave Grossman, asserts that "having PTSD is more like being fat than like having diabetes — you can choose not to be fat."[21]

AN ALTERNATIVE MODEL: STRESS INJURIES AS LITERAL WOUNDS TO THE BRAIN AND MIND

Although traditional conceptions of trauma in psychology have tended to view stress-induced wounds to the mind as more metaphorical than literal, the notion that traumatic experiences can inflict literal wounds to the brain has a long tradition. In contrast to Freud's view that peritraumatic breakdown was a self-protective defense mechanism, his contemporary, Janet, believed that such breakdown was due to a failure of integrative brain functions under the impact of a "vehement emotion."[22] Modern research on the phenomena of dissociation and their neurological basis has revived Janet's viewpoint. Recent neurobiological studies in both humans and animals make a compelling argument that intense or prolonged stress can inflict literal injuries to the brain and other organs of the body.

The strongest evidence that severe stress can inflict literal injuries to the brain comes from studies of glutamatergic pathways in the brains of humans and animals, particularly those in the limbic system and cerebral cortex that use the N-methyl-D-aspartate (NMDA) family of receptors. Glutamate is the primary excitatory neurotransmitter in the brain. NMDA receptors for glutamate play a crucial role in neuronal growth and development, and neuronal plasticity, such as that required for learning and memory.[23] Glutamate neurons bearing NMDA receptors are fundamental to the normal functions of the hippocampus — a sea horse-shaped gray matter structure in each temporal lobe — including:[24]

- Intermediate declarative memory acquisition and retrieval;
- Consolidation of declarative information into long-term storage;
- Extinction of fear conditioning mediated by the amygdala;
- Regulation of neuroendocrine responses to stress;
- Spatial navigation, somewhat like an internal global positioning system (GPS); and
- Possibly social navigation (eg, through recognition of social cues).[25]

Ongoing preclinical and clinical studies of neurobiology are filling in the details in a coherent picture of the damage extreme stress can inflict on the brain.

In all these functions, the hippocampus works in concert with other pathways in the prefrontal cortex that also utilize NMDA receptors for glutamate. For example, extinction of fear-based conditioning is also mediated by the medial prefrontal cortex, and social cue recognition is mediated by the orbitofrontal cortex, in addition to the hippocampus and amygdala. One area of the medial prefrontal cortex that has been increasingly studied in relation to traumatic stress is the anterior cingulate, a brain center essential for the inhibition of situationally inappropriate or irrelevant thoughts and emotions, as well as for the situation-specific regulation of autonomic arousal, including pulse and blood pressure.[26]

Because the symptoms of PTSD prominently include deficits in memory function, the extinction of fear-based learning, authority over one's own emotions and thoughts, and the regulation of autonomic arousal, glutamate neuron systems in the brain are a logical place to look for signs of literal damage from stress. Studies have shown that the same properties of NMDA receptors that make them exceptionally plastic and responsive to the environment also render them vulnerable to damage from excessive stimulation. One of these properties is the unique permeability of NMDA receptors to calcium, which is central to the intracellular sequence of events that can result in neuronal damage or death.[27]

As depicted in the Figure, NMDA receptors in their resting state are impermeable to calcium because their pores are obstructed by bound magnesium ions. Binding of a glutamate molecule to its site on the NMDA receptor causes the magnesium plug to be ejected, opening the pore for an influx of calcium ions. Once in the cytoplasm, calcium ions are taken up by mitochondria, where they may trigger cell death through two very different pathways: apoptosis (programmed cell death) and necrosis. Apoptosis is the normal process of removal and resorption of cells that are senescent or no longer needed, and apoptosis provokes no reactive inflammatory process. Although apoptosis is a normal process, and therefore necessary for the continual turnover of cells throughout the body, apoptosis of glutamate neurons due to excitotoxic accumulations of intracellular calcium is deleterious, causing shifts in the balance between cell loss and regrowth toward a net loss of cell mass. Excessive intramitochondrial calcium can also trigger cell death through necrosis, resulting largely from the interference by calcium with normal mitochondrial metabolism, effectively starving the neuron to death. The death of neurons by necrosis triggers a local inflammatory process. Regardless of the mechanism of death, ruptured glutamate neurons spill their contents of glutamate into local grey matter, which may further accelerate local glutamate neurotransmission and excitotoxicity.

The molecular events leading to apoptosis or necrosis in glutamate neurons have been found in preclinical studies to be the same regardless of what caused the excess of glutamate neurotransmission in the first place. The excitotoxic cascade in glutamate neurons can be initiated by acute or chronic stress,[28] closed head trauma (eg, concussion),[29] ischemia, and seizures. Its pathological consequences include not only total cell death, but also atrophy of dendritic trees and slowed neurogenesis. Glutamate neurotoxicity is a mechanism that may explain the large overlap in symptoms and functional deficits in such seemingly diverse clinical entities as PTSD, depression, and mild traumatic brain injury.

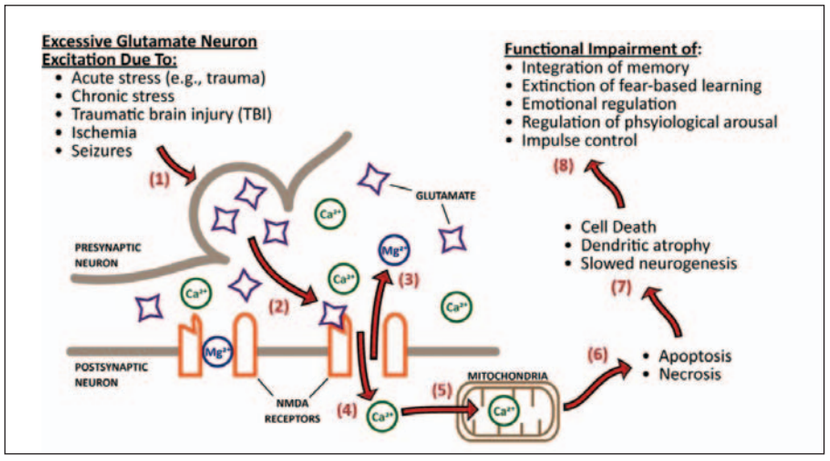

Figure. Excitotoxic cascade leading to damage of glutamate neurons, mediated by N-methyl-D-aspartate (NMDA) receptors. Sequence: (1) glutamate neuron excitation; (2) synaptic release of glutamate and binding to NMDA receptors; (3) removal of Mg^{2+} from pore; (4) influx of CA^{2+}; (5) uptake of CA^{2+} by mitochondria; (6) premature programmed cell death (apoptosis) or necrosis triggered by CA^{2+}; (7) cell death, atrophy, or slowed regrowth; and (8) functional impairment.

Although it is difficult to prove a direct link between molecular or cellular events demonstrated in preclinical studies and clinical pathological states, a number of imaging studies in humans have offered support for the hypothesis that literal damage to glutamate pathways in the hippocampus and prefrontal cortex underlies PTSD, depression, and other stress-related disorders. Multiple imaging studies of adults with depression[24] and PTSD[30] have demonstrated lower hippocampal volumes than controls, although the absence of such findings in children, the lack of prospective studies, and the outcomes of studies of twin pairs discordant for PTSD have raised doubt about the significance of lower hippocampal volumes. More recent volumetric and functional imaging studies of the anterior cingulate cortex suggest a correlation between lower volumes and decreased activity in this region in patients with PTSD.[30]

Ongoing preclinical and clinical studies of neurobiology are filling in the details in a coherent picture of the damage extreme stress can inflict on the brain. Good reasons also exist to conceive of the impacts of trauma and loss on the mind as being literal injuries, as well. Mental function is not infinitely flexible or supported by unrestricted abilities to accurately perceive and assimilate all new information, or to replace all lost attachments with new ones of equal value. Rather, the flexibility and adaptability of mental functions are limited by the enduring mental structures that form the bone and sinew of mind, such as memories, beliefs, assumptions, and attachments.[31] All of these may be damaged in the mind by traumatic experiences or losses no less irreversibly than the body is damaged by physical wounds.

CLINICAL IMPLICATIONS OF THE STRESS INJURY CONCEPTION

The conception of significant distress or functional impairment arising from intense or persistent stress as literal injuries to the brain and mind can significantly reduce the burden of shame associated with the recognition and treatment

of these problems. In clinical care settings, patients and their family members can be greatly relieved to learn that their suffering and functional impairment are not due to their own failure or weakness, any more than any other physical wound would be. When placed in the context of the entire Combat and Operational Stress Continuum — including the four stress "zones" of wellness, mild, and reversible stress reactions, stress injuries, and stress illnesses arising from unhealed stress injuries — this conception can provide a framework for more effective primary and secondary prevention programs in the military and other community settings, as have been adopted recently by the Navy and Marine Corps.[32] By lessening the barriers to early recognition, the stress injury model may also promote more effective and targeted early interventions, such as those based on cognitive-behavioral therapy.[33]

Military leaders and public policy makers may fear that the remedicalization of combat stress and its aftermath may increase the risk for epidemics of stress-injured persons seeking medical evacuation from war zones or disability compensation from the Department of Veterans Affairs. Others may fear that a medical model of stress will increase the burden of stigma borne by service members and veterans by branding them with indelible psychiatric labels. However, it is useful to keep in mind that stigma was attached to mental health labels intentionally as a deterrent to stress-casualty epidemics. There is no reason psychological injuries cannot be stripped of this stigma in the future, and given the same respect as other wounds of war.

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